

# Tuberculous meningitis: role of CT in management and prognosis

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**SUMMARY** Serial computed tomographic scans were performed during the course of tuberculous meningitis in 25 patients aged 1-70 years. Hydrocephalus rarely occurred without other abnormalities. Marked ventricular enlargement was associated with extensive basal enhancement. Basal meningeal enhancement was not a good indicator of the clinical state although marked enhancement was a risk factor for the development of basal ganglia infarction. Infarcts were much more common in children than in adults and were sometimes asymptomatic. Radiological abnormalities sometimes developed during treatment and often did not resolve completely. Many patients had severe residual neurological problems.

Tuberculous meningitis (TBM) is a relatively uncommon condition in the United Kingdom so that few centres have sufficient experience to assess the value of computed tomography (CT) in management. The typical CT appearance of enhancement of the leptomeninges after intravenous contrast has been well documented<sup>1-6</sup> but there is little information regarding the relation of CT changes to symptoms and prognosis, both at the time of the initial study and during follow-up. We have therefore pooled our experience in order to correlate clinical and CT information from a series of 25 cases including both children and adults.

## Materials and methods

There were 12 patients from the London Hospital, 12 from the Hospital for Sick Children, Great Ormond Street and 1 from the National Hospital for Nervous Diseases, Queen Square. In all these patients there were clinical and CSF changes compatible with TBM. All had serial CT scans with a follow up period of more than 1 year except those who died earlier. The patients were classified into three stages of severity according to the Medical Research Council (MRC) criteria of 1948<sup>7</sup> (table 1) and into five stages according to Singhal<sup>8</sup> (table 2). In MRC Stage I category there were six patients, in stage II nine patients and in stage III 10 patients respectively. The patients were also classified according to their clinical state at the end of the follow-up period into

Table 1 *Medical research council staging*

Stage I	Non-specific symptoms. Minimal signs of meningitis no pareses. Fully conscious. Good general condition
Stage II	Condition between stages I and II
Stage III	Extremely ill. Deeply stuporose or comatose with gross pareses

Table 2 *Staging according to Singhal*

Stage I	Systemic symptoms without overt meningitis
Stage II	Overt meningitis
Stage III	Fully conscious. Neurological defects
Stage IV	Stuporose, semi-conscious with or without neurological defect
Stage V	Decerebrate or deeply comatose

four Outcome Groups; Group 1: complete recovery (five patients), Group 2: mild to moderate disability (eight patients), Group 3: severe physical and/or mental disability (six patients), in Group 4: deceased (six patients). Those considered to have a severe residual disability included patients with paraplegia, mental retardation and cerebral palsy. The length of history before commencement of anti-tuberculous chemotherapy, the state of the patient on admission, and the time of insertion of an Ommaya reservoir and/or ventricular diversion and the response to anti-tuberculous therapy were noted.

Detailed analysis of each CT scan included the severity of hydrocephalus and response to therapy, the appearance of the basal cisterns on plain CT and after intravenous contrast medium, the changes in the basal cisterns during chemotherapy, the association of tuberculomas with TBM, and the presence or absence of cerebral infarcts. In addition, the clinical stage of the patients at the commencement of therapy was correlated with the outcome.

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## Results

### CLINICAL FEATURES

#### *Length of history before anti-tuberculous therapy was instituted*

In all but two patients who had long histories (7 and 8 months) the time between onset of symptoms and admission varied between 2 days and 3 months (mean 4 weeks). There was no difference in the range or mean length of history between the outcome groups nor between children and adults, although one of the two patients with a long history died and the other is severely disabled.

Eleven patients had clinical evidence of active recent tuberculous disease in other organs; the lungs were involved in seven and the joints in two, spinal disease with psoas abscess was present in one and mediastinal node enlargement in one. Two other patients had family contact with pulmonary TB and one had had pulmonary tuberculosis 17 years previously. Of the five patients with miliary pulmonary tuberculosis on admission, four had MRC stage III and one MRC stage II TBM; in all but one of these five patients the outcome was poor (Group 3 disability).

Acid fast bacilli were demonstrated in the CSF or by tissue biopsy in 15 patients, three with MRC stage I, four with MRC stage II and eight with MRC stage III disease representing one of five in Outcome Group I, five of seven in Outcome Group II, four of seven in Outcome Group III, (a further two had miliary pulmonary tuberculosis) and five of six in Outcome Group IV. In the remainder the CSF changes were characteristic of tuberculous meningitis.<sup>5 8 9-13</sup>

#### *Morbidity and outcome*

The stage of the disease on admission was correlated

Table 3 Comparison between severity of disease on admission (Singhal and MRC staging) and outcome. Patients identified individually by severity of residual disability at end of follow-up (arabic numerals — see text).

MRC Staging	I	II	III	IV	V
Singhal Staging					
I	1	22	211		
II		21	3	421	234
III			4	423	334

with outcome (table 3). In general, patients with mild disease on admission had a good outcome while those with severe disease did badly. Four children admitted with MRC stage II disease recovered, but no adult admitted with this severity of disease did well. Those with MRC stage III disease remained severely disabled. All patients who ultimately recovered fully (Outcome Group I) responded quickly to anti-tuberculous treatment, three within 1 week and the others within 3 weeks. Patients in Outcome Group II showed a slower response and those in Outcome Group III had a more protracted illness measured in months rather than weeks. Two of the Outcome Group IV patients had a fluctuating but eventually downhill course, death occurring at 8 months and 8 years respectively; two died within a month of presentation and two recovered to a stable state but died suddenly 6 months and 2 years later.

### CT APPEARANCES

#### *Ventricular size*

Hydrocephalus was classified as absent, mild, moderate or severe. Some degree of hydrocephalus was present on admission in 18 patients and developed later in a further three patients. On admission no patients with MRC stage I disease had more than minimal hydrocephalus; four out of nine with MRC stage II and four out of 10 with MRC stage III disease had mild or moderate hydrocephalus. However, three patients with MRC stage II disease and one with MRC stage III disease had normal sized ventricles at presentation but in three of these mild hydrocephalus developed during the first 6 months of treatment. Six of 10 patients with mild hydrocephalus and two of seven with moderate hydrocephalus on admission developed further ventricular enlargement within 2 months.

*Comment* In both adults and children hydrocephalus was commonly present on admission and tended to become more marked during treatment. Although larger ventricular size was usually associated with severe disease on presentation, a few severely ill patients had normal sized ventricles.

#### *CSF reservoirs and permanent ventricular drainage*

All but one of the patients admitted with MRC stage I disease were managed without ventricular reservoirs. Reservoirs were inserted in 19 patients for intraventricular chemotherapy, four of whom had normal sized ventricles. Permanent ventricular drainage was subsequently required in nine patients, (three adults and six children) all of whom had developed moderate hydrocephalus.

*Comment* The ventricles became of normal size in all but one patient with the most severe hydrocephalus and even in this patient there was some

improvement. Of the nine patients who required ventricular drainage two originally admitted with mild clinical disease had mild residual disability and six admitted with severe disease died or had marked residual disability.

### *Infarcts*

Cerebral infarcts were found in eight of the 12 children (aged 1½ to 8 years); a solitary right frontal infarct was present on a scan made on admission in an adult aged 50 years. Infarction was thus much more common in children than in adults. In the children the infarcts involved the basal ganglia and internal capsules; in six children they were bilateral. In two children there were additional infarcts in the deep temporal or occipital lobe; one of these children died. Disability in four of these children and in the adult patient was severe. Three children showed features attributed to hypothalamic dysfunction and only one had no residual disability.

Infarction developed or was present in one of three children with MRC Stage I, three of four with MRC Stage II and four of five with MRC Stage III disease. Infarction was detected on admission in three patients and within 2 weeks of the first scan in the others. Moderate enhancement of the basal cisterns was present in eight of the nine patients at the time of detection of infarction by CT scanning. Among patients without infarcts the same degree of enhancement was present or developed in two children and six adults and less marked basal enhancement was noted in one child and two adults, a total of 11 patients. On follow-up one infarct resolved, three became smaller and the remainder did not change.

*Comment* The presence on admission or the subsequent development of cerebral infarction did not obviously affect the outcome. Although the clinical

features of brain infarction contributed to the initial clinical staging of the disease, patients with clinically mild disease on admission did well despite cerebral infarction and patients with moderate or severe disease did badly. Some infarcts were asymptomatic.

### *Meningeal enhancement*

All the 25 patients had initial CT scans with intravenous contrast. The relationship of meningeal enhancement to prognosis and morbidity was assessed in 23 cases in whom several contrasted CT scans were available. There was no enhancement of the meninges on any scan in five patients followed for 7 to 32 months. In three patients aged 15, 22 and 32 years with no enhancement on the first scan and in one patient aged 3 years with only equivocal enhancement, moderate to marked enhancement of the basal meninges developed within 1 month of admission, despite continuous antituberculous therapy. Three patients had mild and 13 moderate meningeal enhancement on admission (fig 1). Patients with MRC Stage I disease had at most mild enhancement although marked enhancement around the chiasm subsequently developed in one. All patients with moderate enhancement had either MRC Stage II or III disease. Nevertheless three patients with MRC Stage II disease and two with MRC Stage III disease had no enhancement on admission and in only one of these did enhancement develop. Cerebral infarction occurred in only one case without, or with mild, enhancement of the meninges. In eight patients moderate enhancement was not associated with infarction, although in seven of these the extent of enhancement became even more marked during the course of the disease. The extent of basal meningeal enhancement decreased during a period of several months in six patients but resolution to a non-enhancing state occurred in only two of these patients, one with mild and one with moderate initial enhancement, in both cases within 1 month of the first CT scan. Persisting enhancement, more extensive than on the initial scan, occurred in a further seven patients during a follow up of 11 to 96 months. One of these remained well and three others made a good social recovery but remained blind.

The most common sites of meningeal enhancement were the chiasmatic and perimesencephalic cisterns, and one or both sylvian fissures. The chiasmatic cistern was involved in eight patients, six of whom either became blind or had poor vision, usually coinciding with maximum enhancement. Features of hypothalamic damage were noted in two cases, with increased appetite in both and sleep reversal in one. An acute reversible brainstem ischaemic episode occurred at the height of the enhancement in one patient. These clinical features appeared to date from

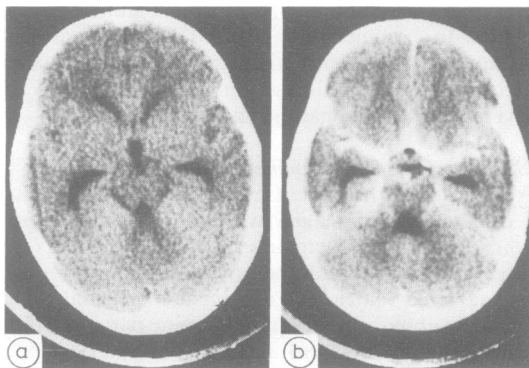


Fig 1 (a) Plain CT scan (b) After contrast injection. There is diffuse enhancement of the basal meninges typical of tuberculous arachnoiditis. Moderate hydrocephalus is also present.

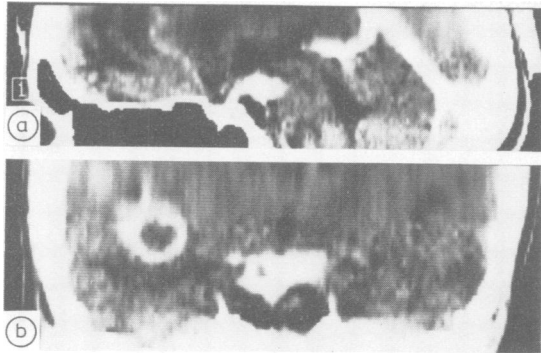


Fig 2 Reformatted (a) sagittal and (b) coronal CT scans after intravenous contrast. There is a dense enhancing mass in the hypothalamus, chiasmatic and infundibular regions associated with a thick walled tuberculoma in the right Sylvian fissure.

the development of marked meningeal enhancement and did not resolve when enhancement became less florid. The sylvian fissures showed selective enhancement in seven patients. Focal seizures occurred in one but no focal features were noted in the other six.

**Comment** Abnormal meningeal enhancement may develop up to 1 month after the initiation of anti-tuberculous therapy. When present it may persist and in many cases did not resolve during the period of observation. Apart from blindness and hypothalamic features which result from chiasmatic and perimesencephalic involvement, meningeal enhancement is not a good indicator of the clinical state. Although marked basal enhancement was a precursor of basal ganglia infarction in our series an equal number of patients with marked enhancement did not develop infarcts.

### Tuberculomas

Tuberculomas occurred in five adults and two children. In four patients they were present on the initial CT scan, and they were the cause of some of the presenting features in all. In the other three patients tuberculomas developed 5 to 7 months after presentation despite antituberculous treatment, and were related to adjacent areas of dense basal enhancement (fig 2). In only one case was resolution achieved on chemotherapy. In a second patient a cerebellar mass was excised completely but in the others, including all who developed granulomatous masses while on treatment, tuberculomas persisted during a follow-up period exceeding 7 months. Two patients had biopsies but this did not modify the CT scan appearance during follow-up. Since they were closely related to areas of dense meningeal enhancement the clinical features of these lesions are discussed above.

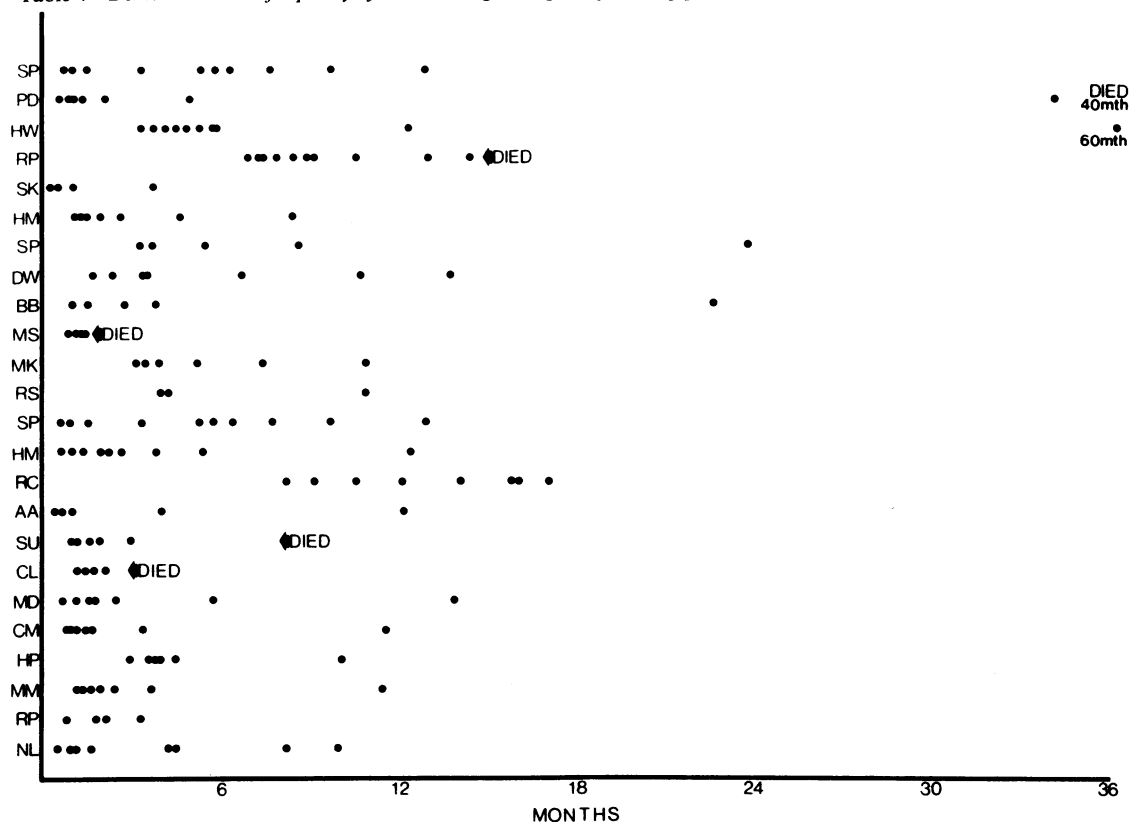
### Discussion

Tuberculous meningitis (TBM) accounts for 45% of all types of tuberculosis among children in India.<sup>5 11-13</sup> According to the MRC tuberculosis survey of England and Wales in 1978/9<sup>7</sup> the overall incidence of TBM is 0.2 per 100,000 in the indigenous white population and 3 per 100,000 in those originating in the Indian sub-continent. As a complication of non respiratory tuberculosis the incidence of TBM is higher in the indigenous white population than in those originating in the Indian sub-continent, and in white children than in white adults.<sup>7</sup>

We have used the MRC clinical staging system in classifying our patients.<sup>3</sup> The system devised by Singhal<sup>4</sup> may however, be more useful since it includes a further sub-division of the MRC Stage II category. We classified the outcome into four grades, Grade 1 indicating full recovery and Grade 4 death, with the two middle grades representing an arbitrary assessment into either mild or severe residual disability, the latter required continuing social management with or without nursing care. Despite the limitations of these crude classifications the two methods chosen corresponded reasonably well (table 3). Comparison of the initial staging and outcome confirms previous reports<sup>3 5 7-9 14 15</sup> that severe disease on admission carries a poor prognosis and mild disease a good prognosis. These findings, however, are qualified in children<sup>16</sup> since in our series several children with severe disease on admission made a complete or near complete recovery. Although CT scanning is important in the diagnosis of TBM<sup>3 5 17-20</sup> there have been few studies of the sequential changes during treatment and their significance in relation to the clinical state.<sup>17-20</sup> The prognosis has usually been correlated only with CT features at diagnosis, thus ignoring those that occur in the course of the disease.<sup>17-19</sup> Our patients were all scanned on admission and in most cases frequently thereafter. Since the length of history before admission varied from 2 days to 8 months, interpretation of this initial CT scan depends not only on the clinical state, but on the rate of evolution of the disease prior to the time it was made.

It is difficult to assess the contribution of hydrocephalus to the state of the patient on admission since it is seldom an isolated abnormality. Hydrocephalus is common in TBM<sup>10 21-24</sup> and is associated with persistent disability and a poor prognosis.<sup>19 22-24</sup> However, in the early stages of TBM ventricular enlargement is not necessarily indicative of progressive disturbance,<sup>10</sup> particularly if treated early.<sup>23</sup> Hydrocephalus alone without other abnormal CT features, such as basal cistern enhancement, is uncommon and in this series only one child and one

Table 4 Demonstrates the frequency of CT scanning throughout follow up period from onset of symptoms.



adult had more than mild hydrocephalus. One responded to repeated tapping of a reservoir and made a complete recovery. The other, in whom diagnosis and treatment had been delayed, had severe hydrocephalus which responded only partially to ventricular drainage leaving a severe neurological disability. Although some clinical improvement followed relief of hydrocephalus, the outcome correlated best with the clinical state on admission. Our experience differs, however, from that of Bhargava *et al*<sup>19</sup> who found a high incidence of hydrocephalus in children. In our series moderate hydrocephalus was found on admission in only four adults and two children and severe hydrocephalus in another two children.

We found a similar incidence of enhancement of the basal cisterns on admission (64%) as previously reported.<sup>3 17-20</sup> This did not change during the period of follow-up in 75% of these patients; complete resolution occurred in only two. It commenced during treatment in four patients, in three of whom marked enhancement was associated with the development of significant hydrocephalus. Severe meningeal enhancement, said to be common in children,<sup>19</sup> was not present in any of our patients on admission

and occurred during the course of treatment in only three adults and one child. Moderate basal enhancement, on the other hand, was present on admission in 75% of the children and 30% of the adults.

Basal enhancement with ventricular enlargement is a feature of advanced tuberculous meningitis,<sup>6</sup> implying a poor prognosis.<sup>5 18</sup> In our series three children and three adults, all severely ill on admission, had moderate hydrocephalus and basal enhancement. Two of the three children made a reasonable recovery, but the other patients either died or remained severely incapacitated. Since basal enhancement is usually most marked around the optic chiasm and in the interpeduncular cistern<sup>4 5 20 25-29</sup> visual loss is a frequent problem; it occurred in nearly 25% in this group of patients. It usually begins about 6 to 8 weeks after the onset of the disease<sup>27 29 30</sup> but may develop much later. Treatment with anti-tuberculous agents does not improve vision and progressive deterioration may occur during treatment,<sup>25 28</sup> probably owing to ischaemic changes.<sup>4 22 26</sup> Despite reports suggesting that surgery may reverse the progression of the visual loss<sup>27 28</sup> no improvement occurred in the two patients in our series who were explored. The timing of the

exploration and the state of the disease may be important factors in the outcome of surgery. During treatment the gelatinous, inflammatory, perichiasmatic exudate resolves to a hard fibrous connective tissue<sup>22 29</sup> which can continue to be laid down even when the meningitis has been cured.<sup>31</sup> However, sometimes granulomatous meningitis persists,<sup>32</sup> eventually becoming dormant and encapsulated, although continuing to pose a potential threat.<sup>33</sup> Treatment of this form of TBM thus remains unsatisfactory.

Tuberculous arachnoiditis frequently involves the basal arteries of the brain leading to endarteritic occlusion involving the perforating arteries.<sup>4 22</sup> This probably accounts for the frequency with which lacunar infarcts involve the basal ganglia.<sup>4 22</sup> Although all but one of the infarcts in our series occurred in children, other reports describe a similar incidence in adults.<sup>10-13 22 34</sup> Infarction may be preceded by the early occurrence of diffuse basal oedema involving the subthalamic or subputaminal areas<sup>16 22</sup> a feature not encountered in our patients.

Cerebral tuberculomas are frequently found in patients dying of meningitis.<sup>20 22</sup> Before CT scanning became widely available they were considered to be rare in successfully treated tuberculous meningitis.<sup>13 30</sup> However, more recent experience indicates that they are not uncommon.<sup>5 35 36</sup> Our experience supports this view and indicates that although tuberculous masses may be present on admission they may also develop during apparently successful anti-tuberculous chemotherapy.<sup>5 35 36</sup> They are usually located close to the surface of the brain and often develop in an area adjacent to florid meningeal enhancement.<sup>5</sup>

During the convalescent phase a number of clinical features have been recorded which may result from damage to the hypothalamus. These include precocious puberty,<sup>37 38</sup> diabetes insipidus,<sup>9 37 38</sup> Cushing's syndrome<sup>38</sup> and severe obesity.<sup>9 38</sup> Two of the patients in this series developed voracious appetites<sup>9</sup> and one a reversed sleep pattern.<sup>9</sup> These patients had infarcts in the basal ganglia and marked basal meningeal enhancement suggesting that hypothalamic infarction had resulted from endarteritis.<sup>22</sup>

Despite the availability of anti-tuberculous chemotherapy the morbidity remains high with a mortality of up to 38%.<sup>11-13 30 39</sup> Twenty five percent of our patients died. Only half of these deaths were a direct result of TBM, intercurrent infection being an important and potentially fatal complication in disabled patients. The reported incidence of severe residual sequelae is 35% to 53%.<sup>5 9 14 16 37 40</sup> consisting of cranial nerve palsies, decerebration, hemiparesis or paraparesis, epilepsy and intellectual deficits. One of the most important prognostic factors

is delay between the onset of symptoms and the initiation of anti-tuberculous therapy.<sup>9 16 21 30</sup> Seven of our patients remained untreated for periods exceeding 8 weeks; one of these died, four remained severely disabled, and none made a complete recovery. All but one had serious disease according to the classification of Singhal.<sup>19</sup>

## Conclusions

Our study shows that CT is valuable in the management of patients with tuberculous meningitis. It should be undertaken during initial investigation in order to provide baseline data and to detect abnormalities, especially hydrocephalus, which may require more active intervention. Hydrocephalus alone is uncommon and has a good prognosis. It is often associated with basal enhancement and in our series became more severe with increasing basal enhancement. Hydrocephalus, enhancement of the basal meninges and tuberculomas may develop during treatment and often do not resolve completely. Contrary to the experience of others infarcts were commoner in children in our series and were sometimes asymptomatic. Cerebral infarcts did not appear to alter the prognosis but did contribute to the staging of the disease on admission. The prognosis of tuberculous meningitis remains poor, over 50% either dying or remaining severely incapacitated.

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